REMARKS

I. Election/Restrictions

Claims 1-3 and 8 have been withdrawn, without prejudice, as being drawn to a non-elected invention.

II. Claim Rejections – 35 U.S.C. 112, Second Paragraph

Claims 4-7 and 9 were rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The Examiner states that claim 4 is confusing on the basis that it recites that the patient is suffering from diabetic cardiomyopathy in the preamble but does not do this in the body of the claim. Although Applicants do not believe this claim is confusing, claim 4 has now been amended to include this recitation in the claim body, thereby alleviating this ground of rejection.

III. Claim Rejections - 35 U.S.C. 103

A. Aspnes or Du Bois in View of Horikawa

Claims 4-7 and 9 were rejected under 35 U.S.C. 103(a) as being unpatentable over Aspnes et al. (col. 15) or Du Bois (col. 24) in view of Horikawa et al. (col. 13). The Examiner argues that it would have been obvious to one of ordinary skill in the art to treat someone with diabetic cardiomyopathy with GLP-1 since it is clear from Horikawa that diabetic patients often have conditions such as cardiomyopathy and since Aspnes and Du Bois teach that diabetics can be treated using GLP-1 it would have been obvious to treat diabetic cardiomyopathy with GLP-1. Applicant respectfully traverses this rejection.

The PTO bears the burden of establishing a case of prima facie obviousness. In re Fine, 837 F.2d 1071, 1074 (Fed. Cir. 1988). It is axiomatic that in order to establish a prima facie case

of obviousness, it is necessary for the examiner to present evidence, preferably in the form of some teaching, suggestion, incentive or inference in the applied prior art, that one having ordinary skill in the art would have been led to combine the relevant teachings of the applied references in the proposed manner to arrive at the claimed invention. See e.g. Carella v. Starlight Archery, 804 F.2d 135 (Fed. Cir. 1986); Ashland Oil, Inc. v. Delta Resins & Refractories, Inc., 776 F.2d 281 (Fed. Cir. 1985). This suggestion cannot stem from the applicant's own disclosure, however. In re Ehrreich, 590 F.2d 902 (CCPA 1979).

The Examiner's obviousness rejection is based on the premise that it would have been obvious to treat any of the complications of diabetes with GLP-1 on the basis that it was known to treat diabetes with GLP-1. However, this approach is not consistent with the teachings of the cited references, or treatments for diabetic cardiomyopathy known in the art.

Horikawa discusses the fact that some patients are virtually impossible to treat with insulin because their cells cannot effectively utilize or are resistant to insulin therapy. (Col. 13, lines 45-47). Thus, as a result of the lack of glycemic control, diabetic patients often experience a variety of complications, including neuropathy, nephropathy, cardiomyopathy, fetinopathy, coronary, and peripherovascular disease. (Col. 13, lines 47-50). Horikawa notes that these complications occur "due to the unachieved glycemic control that results from failure of the insulin, diet and/or exercise only approach." (Col. 13, lines 47-51). Thus, while insulin and other substances such as thiazolidinediones, sulfonylureas, and GLP-1 are known to treat diabetes by increasing insulin levels and decreasing glucose levels, some diabetics develop complications such as cardiomyopathy when their insulin levels can not be effectively controlled.

Cardiomyopathy is a condition in which heart muscle dies. (See attached articles). The myocardium becomes inflamed, scarred, and fibrous, and the heart may enlarge and become

inefficient in pumping blood. People with cardiomyopathy may suffer from shortness of breath, tire easily, develop ankle swelling, and suffer from chest pains.

Conventional treatments for cardiomyopathy are used to assist the weakened heart in pumping blood, and include:

-diuretics: to remove excess fluid from the body;

-vasodilators, such as ACE inhibitors, to relax blood vessels and lower blood pressure;

-calcium channel blockers and beta blockers to regulate heartbeat and alter the work of the heart muscle;

-digitalis, to improve pumping action and regulate heartbeat; and

-antiarrhythmics.

Where the condition is severe enough, a heart transplant may be necessary.

While GLP-1, insulin, and other treatments used to maintain diabetics' insulin levels have been known to prevent diabetic cardiomyopathy, it does not follow that a person skilled in the art would have been inclined to use GLP-1 to treat cardiomyopathy, or any of the other complications of diabetes for that matter.[1] As shown above, once cardiomyopathy occurs, current treatments have included conventional heart and blood pressure lowering medications, not drugs for treating diabetes. More particularly, once elevated glucose levels have damaged the heart, the focus of treatment has ordinarily focused on improving the function and reducing the workload of the heart.

[1] Similarly, while control of blood sugar levels can protect against diabetic retinopathy, once it occurs, the focus of treatment shifts to improving damage to the eye, including laser surgery and vitrectomy.

In this case, Applicants have surprisingly discovered that GLP-1 is useful in treating cardiomyopathy once it has occurred in the patient by reducing norepinephrine levels in the heart and/or plasma. The cited references do not suggest or teach that administration of GLP-1 would be useful for this purpose, as set forth in claims 4-7 and 9. The cited references further do not teach administration of GLP-1 molecule in a dose of from about 0.1-10 pmol/kg/min (new claims 10 and 14), subcutaneous administration of GLP-1 in a dose of from about 0.5-50 pmol/kg/min (new claims 11 and 15), administration of GLP-1 in a dose up to about 10.0 nmol/kg (new claims 12-13), or administration of GLP-1 (7-36) amide (new claim 16). Hence, the Examiner has failed to establish a prima facie case of obviousness with respect to Aspnes or Du Bois in view of Horikawa. Applicants therefore respectfully request that this ground of rejection be withdrawn.

B. Efendic in View of Horikawa et al.

Claims 4-7 and 9 were rejected under 35 U.S.C. 103(a) as being unpatentable over Efendic (examples and the claims) in view of Horikawa et al. Again, the Examiner argues that it would have been obvious to one of ordinary skill in the art to treat someone with diabetic cardiomyopathy with GLP-1 since it is clear from Horikawa that diabetic patients often have conditions such as cardiomyopathy and since Efendic teaches that diabetics can be treated using GLP-1 it would have been obvious to treat diabetic cardiomyopathy with GLP-1.

As already shown above, the cited references do not teach persons skilled in the art that GLP-1 or other blood glucose lowering agents may be useful for treating diabetic complications once they have developed. Instead, the only teachings that may possibly be garnered from the references is that agents used for normalizing plasma glucose levels may be useful in the prevention of these complications. Since Efendic and Horikawa do not provide a teaching or suggestion to persons skilled in the art to use GLP-1 for treating a person already suffering from

cardiomyopathy (as claimed), they do not render claims 4-7, 9, and new 10-16 obvious. Applicants therefore respectfully request that this ground of rejection also be withdrawn.

IV. Conclusion

For the above-stated reasons, it is believed the application is in a prima facie condition for allowance. Allowance is respectfully requested.

It is not believed any fees are due with this response. If this is not correct, please consider this a request to credit or debit Deposit Account No. 26-0084.

Attached hereto is a marked-up version of the changes made to the specification and claims by the current amendment. The attached page is captioned "**Version with markings to show changes made.**"

Respectfully submitted,

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AMENDMENT — VERSION WITH MARKINGS TO SHOW CHANGES MADE — DO NOT FILE

In the Claims

Claims 1-3 and 8 have been canceled as being drawn to a non-elected invention.

Claim 4 was amended as follows:

4. (Amended)

A method of treating a patient suffering from diabetic cardiomyopathy, comprising administering a therapeutically effect amount of a GLP-1 molecule to [said] a patient suffering from diabetic cardiomyopathy.

Claims 10-16 were added:

10. (New)

The method of claim 6 whereby the GLP-1 molecule is administered in a dose of from about 0.1-10 pmol/kg/min.

11. (New)

The method of claim 4 whereby the GLP-1 molecule is administered subcutaneously in a dose of from about 0.5-50 pmol/kg/min.

12. (New)

The method of claim 4 whereby the GLP-1 molecule is administered in a dose of up to 10.0 nmol/kg.

13. (New)

A method of treating a patient suffering from diabetic cardiomyopathy, comprising administering up to 10.0 nmol/kg of a GLP-1 molecule to a patient suffering from diabetic cardiomyopathy.

14. (New)

The method of claim 13 whereby the GLP-1 molecule is administered intravenously in a dose of from about 0.1-10 pmol/kg/min.

15. (New)

The method of claim 13 whereby the GLP-1 molecule is administered subcutaneously in a dose of from about 0.5-50 pmol/kg/min.

16. (New)

The method of claim 13 whereby the GLP-1 molecule is GLP-1 (7-36) amide.

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FAQ (Health)
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Dr. George Jacob
Heart Infocenter

Holistic-online.com

Cardiomyopathy

Introduction to Cardiomyopathy

Cardiomyopathy is a condition in which the death of heart muscle results in the ultimate heart failure. The heart muscle tissue, known as the myocardium, becomes inflamed, scarred and fibrous. The walls of the heart may become thick and hard or thin and weak. The heart may enlarge and become inefficient in pumping blood. So it start beating faster, trying to play catch up.

People with cardiomyopathy may suffer from shortness of breath (breathless) when they are active. Sometimes, depending on the severity of their conditions, they may suffer from shortness of breath when they are not doing anything. They may tire easily, develop ankle swelling and may suffer from chest pains.

Compared with coronary heart disease, the most common form of heart disease, cardiomyopathy is rare.

No satisfactory treatment is available to treat cardiomyopathy except for heart transplant. Drugs are useful to provide some relief by reducing demands on the heart. Mind body medicine and nutritional therapy may be useful in stabilizing or even reversing the condition.

Cardiomyopathy can be caused by many known diseases. However, in some cases, it may have no identifiable cause.

Cardiomyopathy can be caused by fat-clogged arteries. But it does not have to be. It may be caused by a virus or another type of infection, such as Lyme disease or AIDS; an inherited metabolic disorder; exposure to toxic chemicals such as cobalt, lead or carbon monoxide; sensitivity to commonly used drugs; toxins such as alcohol or cocaine; or heart damage caused by a disease such as diabetes.

Poor nutrition also may play a role in the development of some forms of cardiomyopathy or in worsening its symptoms.

Depending on the cause, the cardiomyopathy can be classified under:

Dilated Congestive Cardiomyopathy - A group of heart disorders in which the ventricles enlarge but aren't able to pump enough blood for the body's needs, resulting in heart failure.

Hypertrophic Cardiomyopathy - A group of heart disorders in which the walls of the ventricles thicken.

Restrictive Cardiomyopathy - A group of disorders of the heart muscle in which the walls of the ventricles become stiff, but not necessarily thickened, and resist normal filling with blood between heartbeats.

Caution: Cardiomyopathy is a life-threatening condition. If you suspect you or someone you know is suffering from cardiomyopathy, seek medical assistance immediately.

Next Topic:

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Facts About Cardiomyopathy

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INTRODUCTION

Cardiomyopathy is a disease of the heart muscle. The heart loses its ability to pump blood and, in some instances, heart rhythm is disturbed, leading to irregular heartbeats, or arrhythmias. Usually, the exact cause of the muscle damage is never found.

Cardiomyopathy differs from many other heart disorders in a couple of ways. First, the types not related to coronary atherosclerosis are fairly uncommon. Cardiomyopathy affects about 50,000 Americans. However, the condition is a leading reason for heart transplantation.

Second, unlike many other forms of heart disease that affect middle-aged and older persons, certain types of cardiomopathies can, and often do, occur in the young. The condition tends to be progressive and sometimes worsens fairly quickly.

NONISCHEMIC CARDIOMYOPATHY

As noted, there are various types of cardiomyopathy. These fall into two major categories: "ischemic" and "nonischemic" cardiomyopathy.

- Ischemic cardiomyopathy typically refers to heart muscle damage that results from coronary artery disease, such as heart attack, and will not be discussed here (see page 8 on how to get information on the disorder).

- Nonischemic cardiomyopathy includes several types. The three main types are covered in this fact sheet. They are: dilated, hypertrophic, and restrictive. The name of each describes the nature of its muscle damage.

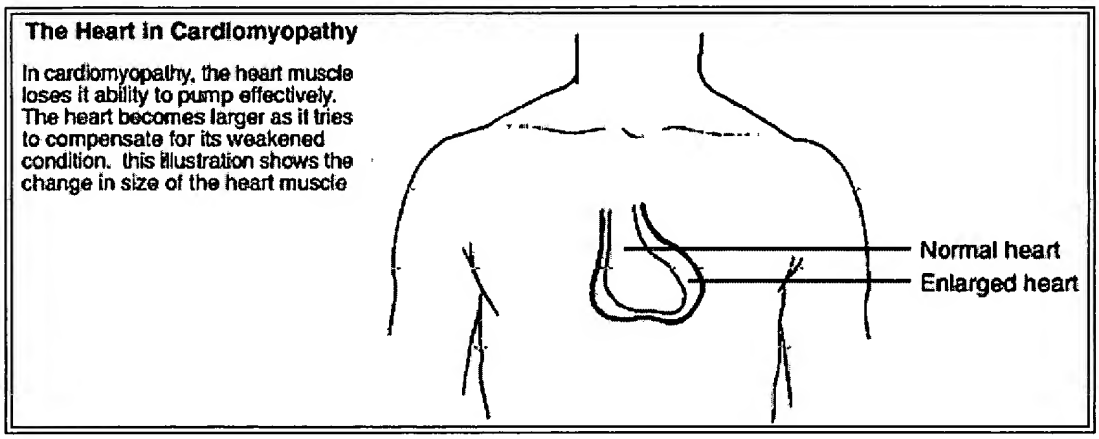
Dilated (Congestive) Cardiomyopathy

By far the most common type of nonischemic cardiomyopathy, the dilated (stretched) form occurs when

disease-affected muscle fibers lead to enlargement, or dilation, of one or more chambers of the heart. This weakens the heart's pumping ability. The heart tries to cope with the pumping limitation by further enlarging and stretching--a process known as "compensation."

Dilated cardiomyopathy occurs most often in middle-aged people and more often in men than women. However, the disease has been diagnosed in people of all ages, including children.

In most cases, the disease is idiopathic--a specific cause for the damage is never identified.



But some factors have been linked to the disease's occurrence. For instance, alcohol has a direct suppressant effect on the heart. Dilated cardiomyopathy can be caused by chronic, excessive consumption of alcohol, particularly in combination with dietary deficiencies. Also, dilated cardiomyopathy occasionally occurs as a complication of pregnancy and childbirth. Other factors are: various infections, mostly viral, which lead to an inflammation of the heart muscle (myocarditis); toxins (such as cobalt, once used in beers, for instance); and, rarely, heredity.

Some drugs, used to treat a different medical condition, also can damage the heart and produce dilated cardiomyopathy. Such drugs include doxorubicin and daunorubicin, both used to treat cancer.

Whatever the cause, the clinical and pathological manifestations of dilated cardiomyopathy are usually the same.

Symptoms

Dilated cardiomyopathy can be present for several years without causing significant symptoms. With time, however, the enlarged heart gradually weakens.

This condition is commonly called "heart failure," and it is the hallmark of dilated cardiomyopathy. Typical signs and symptoms of heart failure include: fatigue; weakness; shortness of breath, sometimes severe and accompanied by a cough, particularly with exertion or when lying down; and swelling of the legs and feet, resulting from fluid accumulation that may also affect the lungs (congestion) and other parts of the body. It also produces abnormal weight gain. (The cough and congestion mimic and, therefore, can be misdiagnosed as pneumonia or acute bronchitis. Also, heart failure is often from heart disease other than cardiomyopathy.)

Because of the congestion, some physicians use the older term "congestive cardiomyopathy" to refer to dilated cardiomyopathy. In advanced stages of the disease, the congestion may cause pain in the chest or abdomen.

In advanced stages, some patients develop irregular heartbeats, which can be serious and even life threatening.

Diagnosis

Once symptoms appear, the condition may be tentatively diagnosed based on a physical examination and a patient's medical history. More often, though, further examination is needed to differentiate dilated cardiomyopathy from other causes of heart failure.

A firm diagnosis typically requires a chest x ray to show whether the heart is enlarged, an electrocardiogram to reveal any abnormal electrical activity of the heart, and an echocardiogram, which uses sound waves to produce pictures of the heart.

Other, more specific tests may also be needed. These include:

- A radionuclide ventriculogram. This involves injecting low-dose radioactive material (usually equal to that in a set of chest x rays) into a vein, through which it flows to the heart. Pictures are generated by a special camera to show how well the heart is functioning.
- A cardiac catheterization. In this procedure, a thin plastic tube is inserted through a blood vessel until it reaches the heart. A dye is injected and x rays taken to assess the heart's structure and function.

Treatment

Since dilated cardiomyopathy is hard to diagnose early, it is rarely treated in its beginning stage.

The goal of treatment is to relieve any complicating factor, if known, control the symptoms, and stop the disease's progression. However, no cure now exists.

Therapy begins with the elimination of obvious risk factors, such as alcohol consumption. Weight loss and dietary changes, especially salt restriction, may also be advised.

Drugs used to treat the condition include:

- Diuretics, which reduce excess fluid in the body;
- Vasodilators, such as angiotensin-converting enzyme (ACE) inhibitors, which relax blood vessels, helping to lower blood pressure and reducing the effort needed by the heart to pump blood through the body;
- Digitalis, which helps to improve pumping action and regulate heartbeat; and,
- Calcium blockers or beta blockers, which may be used in some patients to help regulate heartbeat and to alter the work of the heart muscle.

Also, patients with irregular heartbeats may be put on any of various drugs to control the rhythm.

In critical cases where the condition is advanced and the patient does not sufficiently respond to other treatments, a heart transplantation may be needed. The patient's heart is replaced with a donor heart. Most heart transplant recipients are under age 60 and in good health other than their diseased heart.

Course of the disease

As the heart enlarges, it steadily decreases its efficiency in pumping blood and the amount of blood it can pump. As a result, some patients cannot perform even simple physical activities.

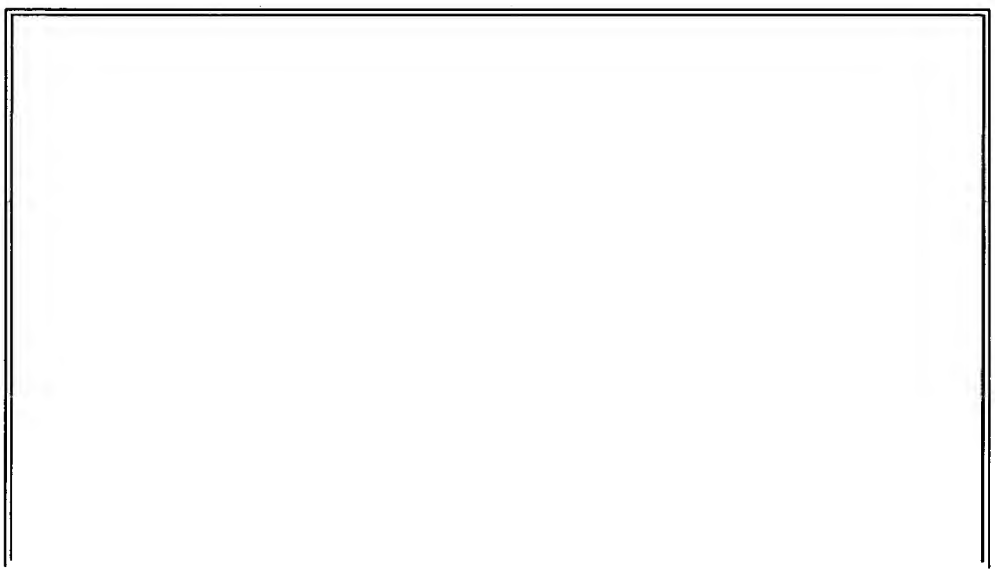
However, the disease also may remain fairly stable for years, especially with treatment and regular evaluation by a physician.

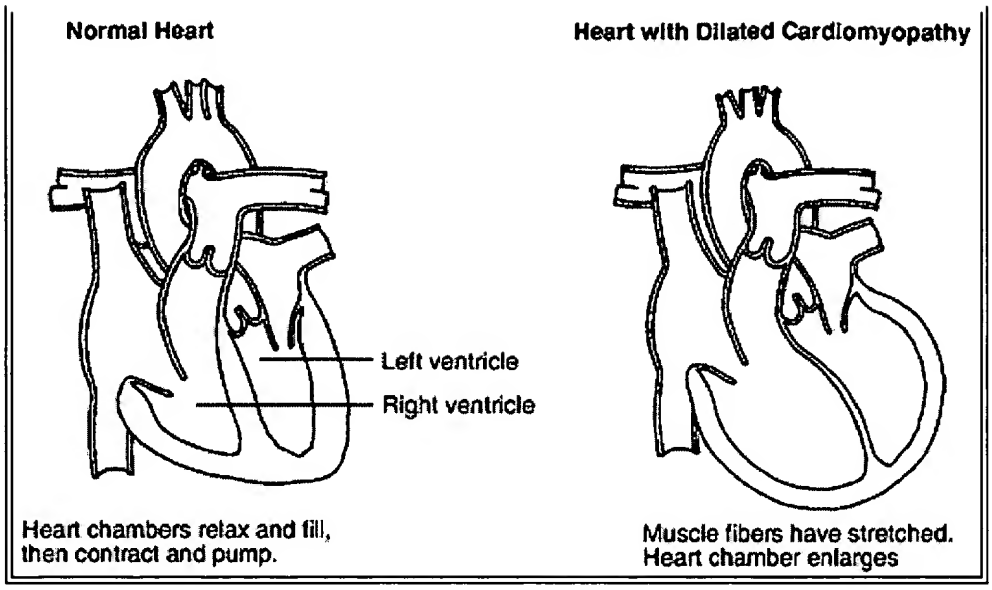
Unfortunately, by the time it is diagnosed, the disease often has reached an advanced stage and heart failure has occurred. Consequently, about 50 percent of patients with dilated cardiomyopathy live 5 years once heart failure is diagnosed; about 25 percent live 10 years after such a diagnosis.

Typically, patients die from a continued decline in heart muscle strength, but some die suddenly of irregular heartbeats.

For patients with advanced disease, heart transplantation greatly improves survival: 75 percent of patients live 5 years after a transplantation. However, in the United States, the scarcity of donor hearts limits the number of transplantations to about 2,000 persons a year. Those who qualify for heart transplantation often have to wait months, or even years, for a suitable donor heart. Some patients with dilated cardiomyopathy die awaiting a transplant but, according to recent studies, others improve enough from aggressive medical treatment to be taken off the waiting list.

Also, some critically ill cardiomyopathy patients with declining heart function use a small, implanted mechanical pump as a bridge to transplantation. Called left ventricular assist devices (LVADs), these pumps take over part or virtually all of the heart's blood pumping activity. The devices provided only temporary assistance and are not now used as substitutes for heart transplantation.





Hypertrophic Cardiomyopathy

The second most common form of heart muscle disease is hypertrophic cardiomyopathy. Physicians sometimes call it by other names: idiopathic hypertrophic subaortic stenosis (IHSS), asymmetrical septal hypertrophy (ASH), or hypertrophic obstructive cardiomyopathy (HOCM).

In hypertrophic cardiomyopathy, the growth and arrangement of muscle fibers are abnormal, leading to thickened heart walls. The greatest thickening tends to occur in the left ventricle (the heart's main pumping chamber), especially in the septum, the wall that separates the left and right ventricles. The thickening reduces the size of the pumping chamber and obstructs blood flow. It also prevents the heart from properly relaxing between beats and so filling with blood. Eventually, this limits the pumping action.

Hypertrophic cardiomyopathy is a rare disease, occurring in no more than 0.2 percent of the U.S. population. It can affect men and women of all ages. Symptoms can appear in childhood or adulthood.

Most cases of hypertrophic cardiomyopathy are inherited. Because of this, a patient's family members often are checked for signs of the disease, although the signs may be much less evident or even absent in them. In other cases, there is no clear cause.

Symptoms

Many patients have no symptoms. For those who do, the most common are breathlessness and chest discomfort. Other signs are fainting during physical activity, strong rapid heartbeats that feel like a pounding in the chest, and fatigue, especially with physical exertion.

In some cases, the first and only manifestation of hypertrophic cardiomyopathy is sudden death, caused by a chaotic heartbeat. The heart's lower chambers beat so chaotically and fast that no blood is pumped. Instead of beating, the heart quivers.

In advanced stages of the disease, patients may have severe heart failure and its associated symptoms, including fluid accumulation or congestion.

Diagnosis

By listening through a stethoscope, a physician may hear the abnormal heart sounds characteristic of hypertrophic cardiomyopathy The electrocardiogram (EKG, or ECG) may help diagnose the condition by detecting changes in the electrical activity of the heart as it beats.

Echocardiography is one of the best tools for diagnosing hypertrophic cardiomyopathy. It uses sound waves to detect the extent of muscle-wall thickening and to assess the status of the heart's functioning.

Physicians also may request radionuclide studies to gather added information about the disease's effect on how the heart is pumping blood.

Other tests that also may provide useful information are the chest x ray, cardiac catheterization, and a heart muscle biopsy.

Treatment

Treatments for hypertrophic cardiomyopathy vary but can include the following:

- Lifestyle changes. Patients with serious electrical and blood-flow abnormalities must be less physically active.

- Medications. Various drugs are used to treat the disease. They include beta blockers (to ease symptoms by slowing the heart's pumping action), calcium channel blockers (to relax the heart and reduce the blood pressure in it), antiarrhythmic medications, and diuretics (to ease heart failure symptoms).

  However, drugs do not work in all cases or may cause adverse side effects, such as fluid in the lungs, very low blood pressure, and sudden death. Then, other treatment, such as a pacemaker or surgery, may be needed.

- Pacemakers. These change the pattern and decrease the force of the heart's contractions. The pacemaker can reduce the degree of obstruction and so relieve symptoms. A pacemaker needs to be carefully monitored after its insertion in order to properly adjust the electrical impulse. Some patients who have a pacemaker inserted feel no relief and go on to have heart surgery.

- Surgery. This usually calls for removal of part of the thickened septum (the muscle wall separating the chambers) that is blocking the blood flow. Sometimes, surgery also must replace a heart valve--the mitral valve, which connects the left ventricle and the left atrium, the upper chamber that receives oxygen-rich blood from the lungs.

  Surgery to remove the thickening eases symptoms in about 70 percent of patients but results in death in about 1 to 3 percent of patients. Also, about 5 percent of those who have surgery develop a slow heartbeat, which is then corrected with a pacemaker.

Course of the disease

The course of the disease varies. Many patients remain stable; some improve; some worsen in symptoms and lead severely restricted lives. Patients may need drug treatment and careful medical supervision for the rest of their lives.

Hypertrophic cardiomyopathy patients also are at risk of sudden death. About 2 to 3 percent die each year because the heart suddenly stops beating. This cardiac arrest is brought on by an abnormal heartbeat. Over 10 years, the risk of sudden death can be 20 percent or more.

Restrictive Cardiomyopathy

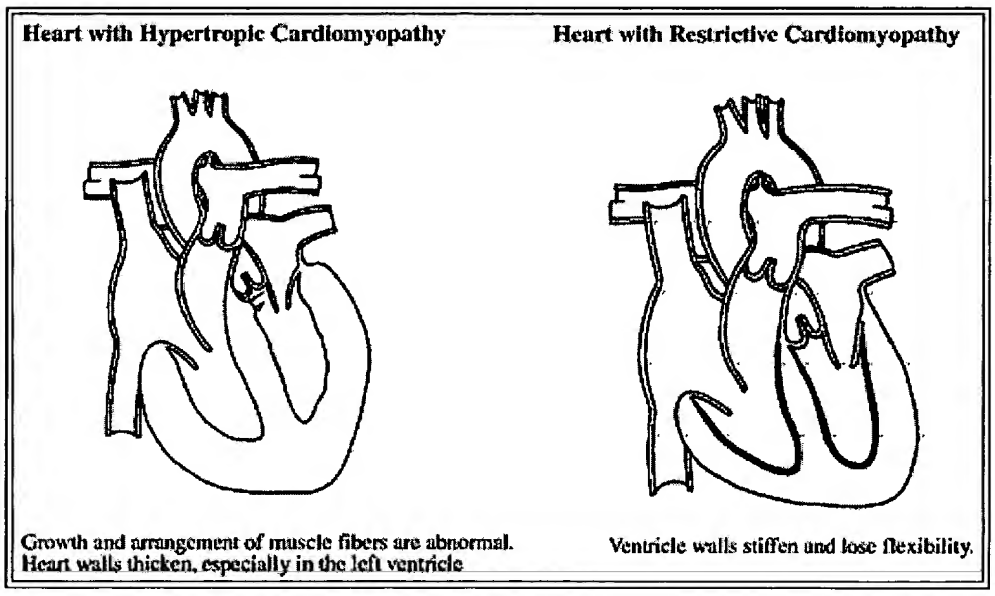
Restrictive cardiomyopathy is rare in the United States and most other industrial nations. In this disease, the walls of the ventricles stiffen and lose their flexibility due to infiltration by abnormal tissue. As a result, the heart cannot fill adequately with blood and eventually loses its ability to pump properly.

Restrictive cardiomyopathy typically results from another disease, which occurs elsewhere in the body. In the United States, restrictive cardiomyopathy is most commonly related to the following: amyloidosis, in which abnormal protein fibers (amyloid) accumulate in the heart's muscle; sarcoidosis, an inflammatory disease that causes the formation of small lumps in organs; and hemochromatosis, an iron overload of the body, usually due to a genetic disease.

In general, restrictive cardiomyopathy does not appear to be inherited; however, some of the diseases that lead to the condition are genetically transmitted.

Symptoms

Typical signs of the condition include symptoms of congestive heart failure: weakness, fatigue, and breathlessness. Swelling of the legs, caused by fluid retention, occurs in a significant number of patients. Other symptoms include nausea, bloating, and poor appetite, probably because of the retention of fluid around the liver, stomach, and intestines.



Diagnosis

A physician may suspect restrictive cardiomyopathy based on a patient's symptoms and the presence of another disease. Although symptoms of congestive heart failure may predominate, the size of the heart remains relatively small, unlike other cardiomyopathies.

Diagnostic information comes from an electrocardiogram or any of several imaging studies that provide pictures of the heart. These include echocardiography, magnetic resonance imaging, and computed tomography.

A definite diagnosis usually requires cardiac catheterization studies or a biopsy, in which a tiny piece of tissue--including heart muscle--is removed for laboratory analysis.

Treatment

Restrictive cardiomyopathy has no specific treatment. The underlying disease that leads to the heart problem also may not be treatable.

In general, the use of traditional heart drugs has been limited in this cardiomyopathy, although diuretics may help control fluid accumulation.

In rare cases, surgery is sometimes used to try to improve blood flow into the heart.

Course of the disease

The condition is similar to dilated cardiomyopathy and tends to worsen with time. Only about 30 percent of patients survive more than 5 years after diagnosis.

FUTURE DIRECTIONS

Future advances in the diagnosis and treatment of cardiomyopathy depend on a better understanding of the disease process and why heart muscle is damaged. A lot of research is under way to identify these processes and whether they can be halted or even reversed. Much of the research is conducted at or supported by the National Heart, Lung, and Blood Institute (NHLBI).

Promising clues came from investigators at and supported by the NHLBI who discovered some of the genes responsible for hypertrophic cardiomyopathy. Their work represents an important first step in understanding how the disease is transmitted and how it progresses.

Researchers also are trying to determine the best use of currently available treatments, especially drug therapies. Drugs useful for other conditions may help treat cardiomyopathy. For example, drugs effective in treating high blood pressure also help manage heart failure and irregular heartbeats.

Additionally, much work has been--and continues to be--done on identifying factors that increase or decrease the risk of death for persons with cardiomyopathy. Knowing which patients are at the greatest risk is very important in determining the best approach to evaluation and treatment of their condition.

The development of improved treatments for cardiomyopathy, however, awaits still more research and a better understanding of the disease process.

GLOSSARY

Angiotensin converting enzyme (ACE) inhibitor--A drug used to decrease pressure inside blood vessels.

Arrhythmia--An irregular heartbeat.

Beta blocker--A drug used to slow the heart rate and reduce pressure inside blood vessels. It also can regulate heart rhythm.

Calcium channel blocker (or calcium blocker)--A drug used to relax the blood vessel and heart muscle, causing pressure inside blood vessels to drop. It also can regulate heart rhythm.

Cardiac arrest--A sudden stop of heart function. See also "sudden death."

Cardiac catheterization--A procedure in which a thin, hollow tube is inserted into a blood vessel. The tube is then advanced through the vessel into the heart, enabling a physician to study the heart and its pumping activity.

Cardiomyopathy--A disease of the heart muscle (myocardium).

Congestion--Abnormal fluid accumulation in the body, especially the lungs.

Digitalis--A drug used to increase the force of the heart's contraction and to regulate specific irregularities of heart rhythm.

Dilated cardiomyopathy--Heart muscle disease that leads to enlargement of the heart's chambers, robbing the heart of its pumping ability.

Diuretic--A drug that helps eliminate excess body fluid; usually used in the treatment of high blood pressure and heart failure.

Dyspnea--Shortness of breath.

Echocardiography--A test that bounces sound waves off the heart to produce pictures of its internal structures.

Edema--Abnormal fluid accumulation in body tissues.

Electrocardiogram (EKG or ECG)--Measurement of electrical activity during heartbeats.

Heart failure--Loss of pumping ability by the heart, often accompanied by fatigue, breathlessness, and excess fluid accumulation in body tissues.

Hypertrophic cardiomyopathy--Heart muscle disease that leads to thickening of the heart walls, interfering with the heart's ability to fill with and pump blood.

Idiopathic--Results from an unknown cause.

Left ventricular assist device (LVAD)--A mechanical device used to increase the heart's pumping ability.

Pulmonary congestion (or edema)--Fluid accumulation in the lungs.

Restrictive cardiomyopathy--Heart muscle disease in which the muscle walls become stiff and lose their flexibility.

Septum--In the heart, a muscle wall separating the chambers.

Sudden death--Cardiac arrest caused by an irregular heartbeat. The term "death" is somewhat misleading, because some patients survive.

Ventricles--The two lower chambers of the heart. The left ventricle is the main pumping chamber in the heart.

Ventricular fibrillation--Rapid, irregular quivering of the heart's ventricles, with no effective heartbeat.

FOR MORE INFORMATION

For more information, contact the NHLBI Information Center, a service of the NHLBI and the National Institutes of Health. The Information Center provides information to health professionals, patients, and the public about the treatment, diagnosis, and prevention of heart, lung, and blood diseases.

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Or check the NHLBI site on the World Wide Web at:
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U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Public Health Service
National Institutes of Health
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NIH Publication No. 97-3082
Originally printed 1995
Revised July 1997

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